a higher intake is required to compare neonatal brain Hg, a better biomarker, across species (Burbacher et al. 1990). However, the present study showed that, at lower MeHg doses, exposure to Hg vapor increased both brain organic and inorganic Hg levels. In addition, the increase in brain Hg did not depend on the Hg vapor dose. That means that brain Hg levels might be higher than expected even if MeHg intake is lower than the provisional tolerance weekly intake or U.S. EPA reference dose when fetuses are simultaneously exposed to Hg vapor even at levels as low as those attributable to dental amalgams. This might be one mechanism by which coexposure to dietary MeHg and Hg vapor at levels relevant to human exposure elevates neurotoxic risks and may need to be taken into account for risk assessment calculations. Additional research is required to directly evaluate such outcomes.

Conclusions

Our study has revealed interactive effects of joint exposure to MeHg and Hg vapor during the prenatal period on organic and inorganic Hg levels in pup brain. Hg vapor increased both forms of Hg in pup brain at lower MeHg concentrations, an outcome relevant to human exposure. Human fetuses exposed to both MeHg and Hg vapor may have increased risks of neurodevelopmental toxicity in contrast to either type of Hg alone.

CORRECTION

In the manuscript originally published online, right-hand columns showing higher Hg vapor doses were inadvertently omitted from Tables 2–5, error bars were omitted from Figure 1A and 1B, and Figure 2B was incorrect. The tables and figures have been corrected here.

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